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Parental smoking and persistent otitis media with effusion in children

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Abstract

A total of 163 children were entered into a case-control study to determine whether any causal relationship exists between otitis media with effusion (OME) requiring grommet insertion and parental smoking. One hundred children with persistent OME formed the case group and 63 children with normal ears formed the control group. The prevalence of parental smoking in each group was then compared. Information was collected by questionnaire and further details about the subjects with regard to surgery of the upper respiratory tract were also gathered. Analysis of findings in this study and previous reports has failed to demonstrate a significantly increased prevalence of smoking in at least one parent, amongst children with persistent otitis media with effusion requiring surgical intervention.

Introduction

Otitis media with effusion is one of the most frequently encountered morbid conditions of childhood. It has a peak incidence between the ages of 3 and 6 years, occurring in 3.6% of 5-year-olds [17]. Up to 80% of all children may experience one episode by 5 years of age [14] with 5–10% requiring surgical intervention.

The condition is important because of its associated conductive hearing loss and consequent impairment of speech and educational progress [2]. It may also be a

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precursor for chronic suppurative otitis media [11,12,14]. In those children requiring operation there are the further attendant risks of surgery and anaesthesia.

Many aetiological factors have been proposed for the development of OME [15]. Eustachian tube dysfunction is considered the final common pathway, contributed to by upper respiratory tract infection [8], local environment [9] and heredity [10].

Recent reports have implicated passive smoking as another predisposing factor in the pathogenesis of OME [3,4,13].

Materials and methods

Information was collected on 100 children at St. George's Hospital, all of whom had surgically proven otitis media with effusion at the time of grommet insertion. Our indication for tympanostomy tubes in this group was the presence of bilateral OME for more than 3 months.

Sixty-three paediatric in-patients attending for orthopaedic or general surgical operations formed a control group. None of these children had existing or previous middle ear pathology. All 163 subjects were referred from within the same Health District and so represent the same range of primary health care assessments before being considered for hospital referral.

Data was compiled on questionnaires. For each of the two groups the prevalence of parental smoking was determined and compared in a standard case-control study.

Subjects in the group with OME were also asked if they had had previous sets of grommets and this was then also related to parental smoking habits. A history of sibling tympanostomy tube insertion amongst this group of children was collected.

The study also examined the relationship of tonsillectomy and adenoidectomy to parental smoking amongst all 163 subjects. Tonsillectomy had been performed for recurrent tonsillitis. Adenoidectomy was performed in children with nasal obstruction, in the absence of nasal mucosal disease, and only after digital and visual examination of the postnasal space under anaesthetic confirmed hypertrophy. The children were therefore separated into new groups, namely operation-by procedure or non-operation. The prevalence of parental smoking in children requiring surgery to the adenoid or tonsil was then compared with that in children not requiring such surgery.

Results

All 163 children came from similar urban areas and social class. Fifty-three (53%) subjects in the OME group and 34 (54%) in the normal ears control group were male. Ages ranged from 2 to 12 (mean 6.01, S.D. 1.9) in the former group and 2 to 10 (mean 6.5, S.D. 2.5) in the latter.

Tonsillectomy and Adenoidectomy

The association of these procedures with parental smoking is demonstrated in Table II. No significant difference in the prevalence of parental smoking amongst the children who required tonsillectomy and the children not requiring this procedure, could be demonstrated (χ^2 with Yates' continuity correction = 0.7 df = 1. $P > 0.5$).

The 30 children undergoing adenoidectomy were also having grommets inserted for persistent OME. A greater proportion of children requiring adenoid removal had non-smoking as opposed to smoking parents.

Discussion

Passive smoking has already been implicated as causing an increase in respiratory illness during infancy and childhood [1,16]. Some authors have further suggested that OME is another direct hazard of parental smoking [3–5,13,15,18]. Studies demonstrating raised salivary cotinine levels in children proportional to their passive tobacco smoke exposure have been seen as complementing these findings [6,7].

It is argued that parental smoking might predispose to OME, not only by increasing upper respiratory tract infections but also by directly irritating the middle ear and eustachian tube mucosa and impairing mucociliary clearance. These actions may be augmented by antigenic substances in tobacco smoke stimulating the adenoid pad to release mediators of inflammation.

Our findings have failed to show any significantly increased prevalence of parental smoking amongst children with persistent OME, than amongst children with normal ears and no history of middle ear pathology. Correspondingly therefore, parental smoking does not result in a significantly increased risk of persistent OME in their offspring. In four previous studies on children in the general population [5,13,15,18] and two on children attending hospital [3,4], only that by Iversen et al. [5] found a significant association between middle ear effusion and passive smoking.

The groups we have studied were both drawn from an in-hospital population. In concentrating on patients requiring surgical intervention for OME, we have specifically looked for a relationship between effusions which are persistent, and parental smoking. It is possible that the prevalence of smoking amongst parents in the control group of children with normal ears may be higher than amongst children with normal ears generally, who do not require general surgical or orthopaedic treatment. However, this is likely to be a small difference only and given the high P values calculated would not produce any significant bias.

Our results were produced from a case-control study examining the prevalence of parental smoking amongst two groups of children. Data on smoking, whilst collected at one moment in time, must invariably be used to represent previous practice. However, none of the parents included had changed their smoking habit during the lifespan of their children at the time of questioning.

Hinton [3] studied children undergoing grommet insertion and compared them with a control group from an orthoptic clinic. He suggested there was a significantly higher than expected prevalence of smoking amongst the parents of children admitted for middle ear ventilation. Information was also collected on 26 children attending the ENT out-patient clinic. If the children from this clinic are also included for analysis, no significant relationship between surgical intervention for OME and passive smoking is demonstrated ($\chi^2 = 2.13$, $0.1 < P < 0.25$).

Strachan et al. [13] related salivary cotinine levels to the prevalence of middle ear effusions in primary school children. Only a third of the cases with flat tympanograms were statistically attributable to passive smoking. Unfortunately no conclusions about persistence of disease may be made from this prevalence study and the survey probably included only a few long standing cases. No information was given on the number of children awaiting grommets or with a previous history of grommet insertion.

In our study not only was no significant difference demonstrated between parental smoking amongst children with persistent OME and those with normal ears, but also a lower percentage of children requiring repeated sets of grommets had smokers as parents.

No significant increase in the prevalence of parental smoking was discovered in children undergoing tonsillectomy or adenoidectomy. Thus recurrent tonsillitis and symptomatic adenoid hypertrophy have not demonstrated any significant association with passive smoking. We have not then been able to support claims that parental tobacco smoke may contribute to offspring OME by causing adenoid hypertrophy.

Conclusion

There is no statistically significant difference in the prevalence of smoking in the parents of children suffering persistent otitis media with effusion, when compared to that amongst parents of children with normal ears.

Correspondingly therefore, parental smoking has not been shown to significantly increase the chance of children developing otitis media with effusion requiring subsequent tympanostomy tube insertion.

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